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=> s ginkgolide (p) breast

626 GINKGOLIDE

66694 BREAST

L1 3 GINKGOLIDE (P) BREAST

=> d 1-3 ab

L1 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN

AB A review. Recent studies conducted with various mol., cellular and whole animal models have revealed that leaf exts. of Ginkgo biloba may have anticancer (chemopreventive) properties that are related to their antioxidant, anti-angiogenic and gene-regulatory actions. The antioxidant and associated anti-lipoperoxidative effects of Ginkgo exts. appear to involve both their flavonoid and terpenoid constituents. The anti-angiogenic activity of the exts. may involve their antioxidant activity and their ability to inhibit both inducible and endothelial forms of nitric oxide synthase. With regard to gene expression, a Ginkgo extract and one of its terpenoid constituents, ginkgolide B, inhibited the proliferation of a highly aggressive human breast cancer cell line and xenografts of this cell line in nude mice. CDNA microarray analyses have shown that exposure of human breast cancer cells to a Ginkgo extract altered the expression of genes that are involved in the regulation of cell proliferation, cell differentiation or apoptosis, and that exposure of human bladder cancer cells to a Ginkgo extract produced an adaptive transcriptional response that augments antioxidant status and inhibits DNA damage. In humans, Ginkgo exts. inhibit the formation of radiation-induced (chromosome-damaging) clastogenic factors and UV light-induced oxidative stress - effects that may also be associated with anticancer activity. Flavonoid and terpenoid constituents of Ginkgo exts. may act in a complementary manner to inhibit several carcinogenesis-related processes, and therefore the total exts. may be required for producing optimal effects.

L1 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN

AB The present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated Ginkgolide B (GKB), a component of the extract of Ginkgo biloba leaves in a method for decreasing the expression of

peripheral-type benzodiazepine receptor (PBR) in cells of a patient in need thereof. Further, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in a method for decreasing the proliferation of cancer cells in a patient. More particularly, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in a method of decreasing cancer cell proliferation in a patient wherein the cancer cell is human breast cancer cell. Even more particularly, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in method of decreasing cancer cell proliferation in a patient wherein the cancer cell is of the aggressive and invasive phenotype and expresses high levels of PBR in comparison to non-aggressive cancer cell.

L1 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN  
AB The peripheral-type benzodiazepine receptor (PBR) expression and localization correlate with human breast cancer cell proliferation and aggressive phenotype expression. The standardized extract of Ginkgo biloba leaves (EGb 761) and isolated ginkgolide B (GKB) were shown to decrease PBR mRNA expression in adrenal cells. The authors examined the effect of EGb 761 and GKB on PBR expression and cell proliferation in human breast cancer cells. EGb 761 and GKB decreased in a time- and dose-dependent manner PBR expression and cell proliferation in the highly aggressive, rich in PBR, human breast cancer cell line MDA-231 whereas they did not affect the proliferation of the non-aggressive human breast cancer cell line MCF-7, which contains very low PBR levels. This effect was reversible and not due to the antioxidant properties of the compds. tested. Using a human cDNA expression array the authors determined that EGb 761 treatment altered, in addition to PBR, the expression of 36 gene products involved in various pathways regulating cell proliferation. These in vitro data were further validated in an in vivo model where EGb 761 and GKB inhibited the nuclear PBR expression and growth of MDA-231 cell xenografts in nude mice. Taken together, these data suggest that the manipulation of PBR expression could be used to control tumor growth and that EGb 761 and GKB, under the conditions used, exert cytostatic properties.

=> d 1-3

L1 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 2003:594452 CAPLUS  
DN 140:35147  
TI Ginkgo biloba extracts and cancer: A research area in its infancy  
AU DeFeudis, Francis V.; Papadopoulos, Vassilios; Drieu, Katy  
CS Institute for BioScience, Westboro, MA, USA  
SO Fundamental & Clinical Pharmacology (2003), 17(4), 405-417  
CODEN: FCPHEZ; ISSN: 0767-3981  
PB Blackwell Publishing Ltd.  
DT Journal; General Review  
LA English  
RE.CNT 106 THERE ARE 106 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 2001:137040 CAPLUS  
DN 134:173024  
TI Ginkgo extract for cancer treatment  
IN Drieu, Katy; Papadopoulos, Vassilios  
PA Societe de Conseils de Recherches et d'Applications Scientifiques, S.A.S., Fr.; Georgetown University  
SO PCT Int. Appl., 50 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001012208	A1	20010222	WO 2000-US22174	20000811
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,				

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CA 2378052 AA 20010222 CA 2000-2378052 20000811  
 EP 1200108 A1 20020502 EP 2000-955489 20000811  
 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, NL, SE, MC, PT,  
 IE, SI, LT, LV, FI, RO, MK, CY, AL  
 JP 2003507336 T2 20030225 JP 2001-516553 20000811  
 NO 2002000666 A 20020211 NO 2002-666 20020211  
 PRAI US 1999-148604P P 19990812  
 WO 2000-US22174 W 20000811

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L1 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN  
 AN 2000:783981 CAPLUS  
 DN 134:320580  
 TI Drug-induced inhibition of the peripheral-type benzodiazepine receptor  
 expression and cell proliferation in human breast cancer cells  
 AU Papadopoulos, Vassilios; Kapsis, Asimina; Li, Hua; Amri, Hakima; Hardwick,  
 Matthew; Culty, Martine; Kasprzyk, Philip G.; Carlson, Mark; Moreau,  
 Jacque-Pierre; Drieu, Katy  
 CS Division of Hormone Research, Georgetown University Medical Center,  
 Washington, DC, 20007, USA  
 SO Anticancer Research (2000), 20(5A), 2835-2847  
 CODEN: ANTRD4; ISSN: 0250-7005  
 PB International Institute of Anticancer Research  
 DT Journal  
 LA English  
 RE.CNT 98 THERE ARE 98 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s ginkgolide (p) benzodiazepine  
 626 GINKGOLIDE  
 20234 BENZODIAZEPINE  
 L2 16 GINKGOLIDE (P) BENZODIAZEPINE  
 => d 1-16 ab

L2 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
 AB The anxiolytic-like effects of Ginkgo biloba extract (GBE) and its four  
 terpenoid components (ginkgolide-A, ginkgolide-B,  
 ginkgolide-C, and bilobalide) were assessed using the elevated  
 plus-maze test in mice. Administration of GBE as a single oral dose (0.5  
 or 1 g/kg, po) caused a state of suppressed motor activity and, thus,  
 shortened the time spent in the open-sided arms. However, when GBE  
 (0.063-1 g/kg, po) was administered daily for 7 days and the plus-maze  
 test was carried out 24 h after the final administration, the time spent  
 in the open-sided arms was prolonged, with the peak anxiolytic-like effect  
 at 0.125 g/kg. A combination of seven-day administration of GBE (0.125  
 g/kg) and a single dose of diazepam (1 mg/kg, po, 10 min before testing)  
 enhanced the anxiolytic-like effect. Flumazenil (0.3 mg/kg, i.p., 10 min  
 before testing) blocked the effect of diazepam, but not of GBE. Daily  
 administration of ginkgolide-A (1 or 2 mg/kg, po) resulted in an  
 anxiolytic-like effect by the third treatment, with the maximal effect  
 observed after the fifth administration. Neither ginkgolide-B,  
 ginkgolide-C, nor bilobalide produced any anxiolytic-like effects.  
 At doses higher than 0.5 g/kg, GBE not only inhibited motor activity but  
 also suppressed active avoidance behavior, reduced caffeine-induced  
 stimulation, and enhanced pentobarbital-induced sleep, while  
 ginkgolide-A (up to 20 mg/kg) did not exhibit these effects.  
 Diazepam (1 mg/kg) is known to enhance pentobarbital-induced sleep. These  
 results suggest that GBE produces a significant anxiolytic-like effect

following repeated administration and that ginkgolide-A is most likely responsible for this effect. There are also indications that although GBE exerts a sedative effect at comparatively higher doses, ginkgolide-A has a relatively weak tendency to produce benzodiazepine-like side effects.

L2 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB Treatment of rats and adrenocortical cells with ginkgolide B (GKB), a purified component of Ginkgo biloba leaf exts., reduces the mRNA, protein, and ligand-binding levels of the adrenal peripheral-type benzodiazepine receptor (PBR), a mitochondrial cholesterol-binding protein, leading to decreased corticosteroid synthesis. In the Y1 adrenocortical cell line, GKB reduced both PBR levels and cAMP-induced steroid formation. In these cells, GKB, but not various steroids and vitamins, reduced the expression of a reporter gene driven by the DNA sequence -624/-513 relative to the transcription start site of the PBR encoding gene. GKB treatment did not affect the SV40 promoter and increased the cytochrome P 450 17 $\alpha$ -hydroxylase gene promoter driven expression of the reporter gene. Electrophoretic mobility shift assays (EMSAs) indicated the presence of a functional transcriptional element bound to the -624/-513 DNA fragment. This GKB-induced inhibition of PBR was mediated by an interaction with a transcription factor that binds to the -636/-616 PBR-promoter region. Deletion or mutation of this sequence eliminated the DNA-protein interaction and the inhibitory effect of GKB on PBR gene transcription. This DNA-binding protein could be detected in nuclear exts. of rat brain, liver, and testis, but not kidney. It is also present in the human adrenal glands. However, the inhibitory effect following GKB treatment could be seen only in the adrenal glands. These results demonstrate that the GKB-activated inhibition of glucocorticoid production is due to a specific transcriptional suppression of the adrenal PBR gene and suggest that GKB might serve as a pharmacol. tool to control excess glucocorticoid formation.

L2 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB A review. Ginkgo biloba leaf extract (EGb 761), which contains many constituents, including flavonoid glycosides and terpenoids (ginkgolides, bilobalide), is used to treat cerebrovascular and peripheral vascular insufficiency, as well as cognitive impairment and other symptoms of dementia. Recent studies have indicated that these therapeutic effects of EGb 761 probably involve modification of the expression of many genes by actions involving several of its active constituents. As examples: EGb 761 and its ginkgolide B constituent inhibit the expression of the peripheral benzodiazepine receptor in the adrenal cortex and decrease circulating levels of corticosterone in the rat, effects that provide a mechanism for explaining the "antistress" action of the extract. Both the flavonoid and terpenoid constituents of EGb 761 decrease the expression of inducible nitric oxide synthase (iNOS), supporting an action of the extract of opposing the deleterious effects of excessive formation of NO. EGb 761 upregulates several genes that encode vital antioxidant enzymes, including heme oxygenase-1 and the regulatory and catalytic subunits of  $\gamma$ -glutamyl-cysteinyl synthetase. Dietary treatment of mice with EGb 761 upregulates the expression of genes encoding neuronal tyrosine/threonine phosphatase 1 and microtubule-associated tau in the cerebral cortex, findings that are of interest since these proteins are associated with the intracellular neurofibrillary tangles found in the brain in Alzheimer's disease. Bilobalide upregulates two mitochondrial-DNA-encoded genes, subunit III of cytochrome c oxidase and subunit ND1 of NADH dehydrogenase, indicating a fundamental mechanism that may underlie EGb 761-induced neuroprotection. Collectively, such results indicate that the therapeutic effects of EGb 761 on cognitive impairment (dementia) may involve its action of altering gene expression.

L2 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB The anxiolytic-like effect of Ginkgo biloba extract (GBE) and its four ginkgo-terpenoids (ginkgolide-A, ginkgolide-B, ginkgolide-C, and bilobalide) were assessed by an improved plus-maze test in mice. The single oral administration of GBE (0.5 and 1 g/kg) shortened the time spent in the open arms with suppression of the motor activity. However, when GBE (0.063-1 g/kg p.o.) was administered

daily for seven days and the plus-maze test was carried out 24 h after the last administration, prolongation of the time spent in the open arms was developed with the peak effect at 0.13 g/kg, showing an anxiolytic-like effect. Diazepam, following the single oral administration at 1 mg/kg, also prolonged the time spent in the open arms. The combination of the seven daily administration of GBE (0.13 g/kg) and single administration of diazepam (1 mg/kg) enhanced the anxiolytic-like effect. Flumazenil (0.3 mg/kg i.p.) blocked the effect of diazepam, but not of GBE. Among the ginkgo-terpenoids, the daily administration of ginkgolide-A (1 and 2 mg/kg p.o.) developed the anxiolytic-like effect by the 3rd administration, and the effect achieved to the highest plateau level by the 5th administration. Whereas, the seven daily treatment with ginkgolide-B (1 mg/kg), ginkgolide-C (1 mg/kg) or bilobalide (1 and 2 mg/kg) caused no anxiolytic-like effect. These results suggest that GBE produces significant anxiolytic-like effect following the repeated administration, and that ginkgolide-A is responsible for this effect. However, it is unlike that benzodiazepine receptors are involved in the development of the anxiolytic-like effect of GBE and ginkgolide-A.

L2 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB Identification of the mol. switch controlling glucocorticoid synthesis might facilitate the development of pharmacol. tools to control circulating cortisol levels. The transport of cholesterol from intracellular sources to the inner mitochondrial membrane represents the rate-determining step in the cascade of reactions leading to cortisol synthesis. A key element in this step is the peripheral-type benzodiazepine receptor (PBR). Several studies have indicated the beneficial effects of Ginkgo biloba on memory and stress control. Using pharmacol., biochem. and proteomic methods, we demonstrated that the standardized Ginkgo biloba extract EGb 761 and its isolated component ginkgolide B (GKB) inhibit PBR ligand binding and protein expression, resulting in decreased serum corticosterone levels. We further demonstrated that EGb 761- and GKB-induced inhibition of PBR protein is preceded by a decrease in mRNA-levels due to transcriptional suppression of PBR gene expression. Further studies indicated that the action of GKB is mediated by a transcription factor binding to the PBR gene promoter, thereby regulating PBR gene expression. These data indicate that EGb 761-induced inhibition of glucocorticoid production is due to specific transcriptional suppression of the adrenal PBR gene by GKB, and suggest that EGb 761 and GKB might serve as pharmacol. tools to control excess glucocorticoid formation.

L2 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB This invention relates to the use of ginkgolides and exts. of Ginkgo biloba for inhibiting the membrane expression of benzodiazepine receptors and for inhibiting the release of glucocorticoids in a patient.

L2 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB The present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated Ginkgolide B (GKB), a component of the extract of Ginkgo biloba leaves in a method for decreasing the expression of peripheral-type benzodiazepine receptor (PBR) in cells of a patient in need thereof. Further, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in a method for decreasing the proliferation of cancer cells in a patient. More particularly, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in a method of decreasing cancer cell proliferation in a patient wherein the cancer cell is human breast cancer cell. Even more particularly, the present invention is directed to the use of the extract of Ginkgo biloba leaves or isolated GKB in method of decreasing cancer cell proliferation in a patient wherein the cancer cell is of the aggressive and invasive phenotype and expresses high levels of PBR in comparison to non-aggressive cancer cell.

L2 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB The peripheral-type benzodiazepine receptor (PBR) expression and localization correlate with human breast cancer cell proliferation and aggressive phenotype expression. The standardized extract of Ginkgo biloba leaves (EGb 761) and isolated ginkgolide B (GKB) were shown to

decrease PBR mRNA expression in adrenal cells. The authors examined the effect of EGb 761 and GKB on PBR expression and cell proliferation in human breast cancer cells. EGb 761 and GKB decreased in a time- and dose-dependent manner PBR expression and cell proliferation in the highly aggressive, rich in PBR, human breast cancer cell line MDA-231 whereas they did not affect the proliferation of the non-aggressive human breast cancer cell line MCF-7, which contains very low PBR levels. This effect was reversible and not due to the antioxidant properties of the compds. tested. Using a human cDNA expression array the authors determined that EGb 761 treatment altered, in addition to PBR, the expression of 36 gene products involved in various pathways regulating cell proliferation. These in vitro data were further validated in an in vivo model where EGb 761 and GKB inhibited the nuclear PBR expression and growth of MDA-231 cell xenografts in nude mice. Taken together, these data suggest that the manipulation of PBR expression could be used to control tumor growth and that EGb 761 and GKB, under the conditions used, exert cytostatic properties.

L2 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB A review with 140 refs. The effects of EGb 761 on the CNS underlie one of its major therapeutic indications; i.e., individuals suffering from deteriorating cerebral mechanisms related to age-associated impairments of memory, attention and other cognitive functions. EGb 761 is currently used as symptomatic treatment for cerebral insufficiency that occurs during normal ageing or which may be due to degenerative dementia, vascular dementia or mixed forms of both, and for neuro-sensory disturbances. Depressive symptoms of patients with Alzheimer's disease (AD) and aged non-Alzheimer patients may also respond to treatment with EGb 761 since this extract has an "anti-stress" effect. Basic and clin. studies, conducted both in vitro and in vivo, support these beneficial neuroprotective effects of EGb 761. EGb 761 has several major actions; it enhances cognition, improves blood rheol. and tissue metabolism, and opposes the detrimental effects of ischemia. Several mechanisms of action are useful in explaining how EGb 761 benefits patients with AD and other age-related, neurodegenerative disorders. In animals, EGb 761 possesses antioxidant and free radical-scavenging activities, it reverses age-related losses in brain  $\alpha$ 1-adrenergic, 5-HT1A and muscarinic receptors, protects against ischemic neuronal death, preserves the function of the hippocampal mossy fiber system, increases hippocampal high-affinity choline uptake, inhibits the down-regulation of hippocampal glucocorticoid receptors, enhances neuronal plasticity, and counteracts the cognitive deficits that follow stress or traumatic brain injury. Identified chemical constituents of EGb 761 have been associated with certain actions. Both flavonoid and ginkgolide constituents are involved in the free radical-scavenging and antioxidant effects of EGb 761 which decrease tissue levels of reactive oxygen species (ROS) and inhibit membrane lipid peroxidn. Regarding EGb 761-induced regulation of cerebral glucose utilization, bilobalide increases the respiratory control ratio of mitochondria by protecting against uncoupling of oxidative phosphorylation, thereby increasing ATP levels, a result that is supported by the finding that bilobalide increases the expression of the mitochondrial DNA-encoded COX III subunit of cytochrome oxidase. With regard to its "anti-stress" effect, EGb 761 acts via its ginkgolide constituents to decrease the expression of the peripheral benzodiazepine receptor (PBR) of the adrenal cortex.

L2 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB In various steroidogenic cell models, mitochondrial preps. and submitochondrial fractions, the expression of the mitochondrial 18 kDa peripheral-type benzodiazepine receptor (PBR) protein confers the ability to take up and release, upon ligand activation, cholesterol. Thus, cholesterol becomes available to P450<sub>scc</sub> on the inner mitochondrial membrane. These in vitro studies were validated by in vivo expts. Treatment of rats with ginkgolide B (GKB), specifically reduced the ligand binding capacity, protein, and mRNA expression of the adrenocortical PBR and circulating glucocorticoid levels. Treatment with GKB also resulted in inhibition of PBR protein synthesis and corticosterone production by isolated adrenocortical cells in response to ACTH. The ontogeny of both PBR binding capacity and protein directly

paralleled that of ACTH-inducible steroidogenesis in rat adrenal cells and in rats injected with ACTH. In addition, the previously described suppression of luteal progesterone synthesis in the pregnant rat by continuous in vivo administration of a gonadotropin-releasing hormone agonist may be due to decreased luteal PBR ligand binding and mRNA. These results suggest that (i) PBR is an absolute prerequisite for adrenocortical and luteal steroidogenesis, (ii) regulation of adrenal PBR expression may be used as a tool to control circulating glucocorticoid levels and (iii) the stress hypo-responsive period of neonatal rats may result from decreased adrenal cortical PBR expression.

L2 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB The hypersecretion of glucocorticoids during exposure to various stressors may induce or worsen pathol. states in predisposed subjects. Therefore it is of interest to evaluate drugs able to reduce glucocorticoid secretion. It has recently been shown that chronic administration of a Ginkgo biloba extract (EGb 761) inhibits stress-induced corticosterone hypersecretion through a reduction in the number of adrenal peripheral benzodiazepine receptors. The present study was designed to analyze the effect of EGb 761 and one of its components, Ginkgolide B on the biosynthesis and secretion of CRH and AVP, the hypothalamic neurohormones that regulate the pituitary-adrenal axis. Chronic administration of EGb 761 (50 or 100 mg/kg p.o. daily for 14 days) reduced basal corticosterone secretion and the subsequent increase in CRH and AVP gene expression. Under the same conditions, surgically-induced increase in CRH secretion was attenuated while the activation of CRH gene expression, ACTH and corticosterone secretion following insulin-induced hypoglycemia remained unchanged. Chronic i.p. injection of Ginkgolide B reduced basal corticosterone secretion without alteration in the subsequent CRH and AVP increase. However, the stimulation of CRH gene expression by insulin-induced hypoglycemia was attenuated by Ginkgolide B. These data confirm that the administration of EGb 761 and Ginkgolide B reduces corticosterone secretion. In addition, these substances act also at the hypothalamic level and are able to reduce CRH expression and secretion. However the latter effect appears to be complex and may depend upon both the nature of stress and substance ( Ginkgolide B or other compds. of EGb 761).

L2 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB It was previously demonstrated that repeated treatment of rats with the standardized extract of Ginkgo biloba leaves, EGb 761, and its bioactive component ginkgolide B (GKB), specifically reduces the ligand binding, and protein and mRNA expression of the adrenal mitochondrial peripheral benzodiazepine receptor (PBR), a key element in the regulation of cholesterol transport, resulting in decreased circulating corticosterone levels. Adrenocortical cells were isolated from rats treated with EGb 761 or GKB and cultured for 2 and 12 days. The effect of ACTH on normal and metabolically labeled cells was examined. Corticosterone levels were measured by RIA, and protein synthesis was analyzed by two-dimensional gel electrophoresis. Ex vivo treatment with EGb 761 and GKB resulted, resp., in 50% and 80% redns. of ACTH-stimulated corticosterone production by adrenocortical cells cultured for 2 days compared with that by cells isolated from saline-treated rats. Two-dimensional gel electrophoresis anal. revealed that in cells from both control and drug-treated animals, ACTH induced the synthesis, at the same level, of a 29-kDa and pI 6.4-6.7 protein identified as the adrenal steroidogenic acute regulatory protein (StAR). In addition, treatment with EGb 761 and GKB specifically altered the synthesis of seven proteins, including inhibition of synthesis of a 17-kDa, identified as PBR. After 12 days in culture, ACTH-stimulated adrenocortical cell steroid synthesis was maintained, and it was identical among the cells isolated from animals treated with GKB or saline. Under the same conditions, the expression of PBR was recovered, whereas no effect of ACTH on the 29-kDa and 6.4-6.7 pI protein (StAR) or other protein synthesis could be seen. A comparative anal. of the effects of GKB and EGb 761 on adrenocortical steroidogenesis and protein synthesis identified, in addition to the 17-kDa PBR, target proteins of 32 kDa (pI 6.7) and 40 kDa (pI 5.7-6.0) as potential mediators of the effect of EGb 761 and GKB on ACTH-stimulated glucocorticoid synthesis. In conclusion, these results (1) validate and extend our previous in vivo findings on the



effect of EGb 761 and GKB on ACTH-stimulated adrenocortical steroidogenesis, (2) demonstrate the specificity and reversibility of EGb 761 and GKB treatment, (3) question the role of the 29-kDa, 6.4-6.7 pI protein (mature StAR) as the sole mediator of ACTH-stimulated steroid production, and (4) demonstrate the obligatory role of PBR in hormone-regulated steroidogenesis.

L2 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB Ginkgolides may be used to inhibit the membrane expression of a benzodiazepine receptor in a patient and in treatments to combat excess glucocorticoid production. A Ginkgo biloba extract, ginkgolide A, and ginkgolide B were tested for their ability to decrease the number of binding sites for the peripheral benzodiazepine receptor ligand PK 11195, which binds to an 18 kDa peripheral benzodiazepine receptor protein in adrenal mitochondria; expression of the receptor was reduced by 40, 50, and 73%, resp. Inhibition of glucocorticoid (e.g. corticosterone) production by ginkgolide A and ginkgolide B is also shown, as well as a corresponding increase in ACTH.

L2 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB Glucocorticoid excess has broad pathogenic potential including neurotoxicity, neuroendangerment, and immunosuppression. Glucocorticoid synthesis is regulated by ACTH, which acts by accelerating the transport of the precursor cholesterol to the mitochondria where steroidogenesis begins. Ginkgo biloba is one of the most ancient trees, and exts. from its leaves have been used in traditional medicine. A standardized extract of Ginkgo biloba leaves, termed EGb 761 (EGb), has been shown to have neuroprotective and antistress effects. In vivo treatment of rats with EGb, and its bioactive components ginkgolide A and B, specifically reduces the ligand binding capacity, protein, and mRNA expression of the adrenocortical mitochondrial peripheral-type benzodiazepine receptor (PBR), a key element in the regulation of cholesterol transport, resulting in decreased corticosteroid synthesis. As expected, the ginkgolide-induced decrease in glucocorticoid levels resulted in increased ACTH release, which in turn induced the expression of the steroidogenic acute regulatory protein. Because ginkgolides reduced the adrenal PBR expression and corticosterone synthesis despite the presence of high levels of steroidogenic acute regulatory protein, these data demonstrate that PBR is indispensable for normal adrenal function. In addition, these results suggest that manipulation of PBR expression could control circulating glucocorticoid levels, and that the antistress and neuroprotective effects of EGb are caused by its effect on glucocorticoid biosynthesis.

L2 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB Pruritis is treated by administration of a therapeutically effective amount of a platelet-activating factor (I) antagonist. The I antagonist may be e.g. a synthetic I analog, a natural product, or a triazolothienodiazepine. I-induced pruritis was blocked by CV-6209 (synthetic I analog), BN 52021 (ginkgolide B), and WEB 2086 (a triazolothienodiazepine derivative), but not by pyrilamine.

L2 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AB Platelet activating factor (PAF) is ubiquitous in mammals, and may have multiple functions in the central nervous system. Triazolobenzodiazepine compds. are active both at the GABAA receptor and as PAF antagonists. To investigate whether PAF antagonist activity is involved in the actions of triazolobenzodiazepines, the effects of two nonbenzodiazepine PAF antagonists on binding and function at the GABAA receptor were investigated. The ginkgolide terpene, BN52021 and the dioxolane-based compound BN 52115 had no effect on benzodiazepine binding or chloride channel binding in cortical membrane prepns. However, chloride uptake into cortical synaptoneurosomes was enhanced with 1 uM BN 52021 but not 1 uM BN 52115. The effect of BN 52021 was prevented by 1 uM flumazenil. PAF antagonists appear to augment GABAA receptor function without affecting binding.

=> d 1-16

L2 ANSWER 1 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 2003:761598 CAPLUS  
DN 139:374862  
TI An Anxiolytic-Like Effect of Ginkgo biloba Extract and Its Constituent,  
Ginkgolide-A, in Mice  
AU Kuribara, Hisashi; Weintraub, Susan T.; Yoshihama, Tatsumi; Maruyama, Yuji  
CS Center for Cooperative Research, Medical Division, Gunma University,  
Showa, Maebashi, Gunma, 371-8511, Japan  
SO Journal of Natural Products (2003), 66(10), 1333-1337  
CODEN: JNPRDF; ISSN: 0163-3864  
PB American Chemical Society  
DT Journal  
LA English  
RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 2003:164618 CAPLUS  
DN 139:79074  
TI Transcriptional suppression of the adrenal cortical peripheral-type  
benzodiazepine receptor gene and inhibition of steroid synthesis  
by ginkgolide B  
AU Amri, Hakima; Drieu, Katy; Papadopoulos, Vassilios  
CS Department of Cell Biology, Division of Hormone Research, Georgetown  
University Medical Center, Washington, DC, 20057, USA  
SO Biochemical Pharmacology (2003), 65(5), 717-729  
CODEN: BCPA6; ISSN: 0006-2952  
PB Elsevier Science Inc.  
DT Journal  
LA English  
RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 2003:122597 CAPLUS  
DN 139:62369  
TI Effects of Ginkgo biloba extract (EGb 761) on gene expression: Possible  
relevance to neurological disorders and age-associated cognitive  
impairment  
AU DeFeudis, Francis V.  
CS Institute for BioScience, Westboro, MA, 01581, USA  
SO Drug Development Research (2002), 57(4), 214-235  
CODEN: DDREDK; ISSN: 0272-4391  
PB Wiley-Liss, Inc.  
DT Journal; General Review  
LA English  
RE.CNT 187 THERE ARE 187 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 2003:33171 CAPLUS  
DN 139:143745  
TI The anxiolytic-like effect of Ginkgo biloba extract and the constituents  
assessed by an improved plus-maze test in mice  
AU Kuribara, Hisashi; Maruyama, Yuji; Yoshihama, Tatsumi  
CS Center for Cooperative Research, Medical Division, Gunma University, Japan  
SO Japanese Pharmacology & Therapeutics (2002), 30(11), 955-962  
CODEN: JPTABU  
PB Raifu Saiensu Shuppan K.K.  
DT Journal  
LA Japanese

L2 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 2002:805581 CAPLUS  
DN 138:314440  
TI Use of ginkgolide B and a ginkgolide-activated  
response element to control gene transcription: Example of the

adrenocortical peripheral-type benzodiazepine receptor  
 AU Amri, Hakima; Drieu, Katy; Papadopoulos, Vassilios  
 CS Institut Henri Beaufour Ipsen, Paris, Fr.  
 SO Cellular and Molecular Biology (Paris, France, Printed) (2002), 48(6),  
 633-639  
 CODEN: CMOBEF; ISSN: 0145-5680  
 PB CMB Association  
 DT Journal  
 LA English  
 RE.CNT 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
 AN 2001:593283 CAPLUS  
 DN 135:157669  
 TI Ginkgolides for inhibition of membrane expression of benzodiazepine  
 receptors  
 IN Drieu, Katy  
 PA Societe de Conseils de Recherches et d'Applications Scientifiques (SCRAS),  
 Fr.  
 SO U.S., 5 pp., Cont.-in-part of U.S. Ser. No. 575,902, abandoned.  
 CODEN: USXXAM  
 DT Patent  
 LA English  
 FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 6274621	B1	20010814	US 1998-68368	19980723
	TW 513305	B	20021211	TW 1996-85113522	19961105
	WO 9717068	A1	19970515	WO 1996-EP5005	19961108
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	ZA 9609437	A	19970618	ZA 1996-9437	19961109
	US 2002006955	A1	20020117	US 2001-879306	20010612
	US 2005281899	A1	20051222	US 2005-130680	20050517
PRAI	US 1995-7337P	P	19951109		
	US 1995-575902	B2	19951220		
	WO 1996-EP5005	W	19961108		
	US 1998-68368	A1	19980723		
	US 2001-879306	A1	20010612		

RE.CNT 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
 AN 2001:137040 CAPLUS  
 DN 134:173024  
 TI Ginkgo extract for cancer treatment  
 IN Drieu, Katy; Papadopoulos, Vassilios  
 PA Societe de Conseils de Recherches et d'Applications Scientifiques, S.A.S.,  
 Fr.; Georgetown University  
 SO PCT Int. Appl., 50 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001012208	A1	20010222	WO 2000-US22174	20000811
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,  
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,  
CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2378052 AA 20010222 CA 2000-2378052 20000811  
EP 1200108 A1 20020502 EP 2000-955489 20000811  
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,  
IE, SI, LT, LV, FI, RO, MK, CY, AL  
JP 2003507336 T2 20030225 JP 2001-516553 20000811  
NO 2002000666 A 20020211 NO 2002-666 20020211  
PRAI US 1999-148604P P 19990812  
WO 2000-US22174 W 20000811

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 2000:783981 CAPLUS  
DN 134:320580

TI Drug-induced inhibition of the peripheral-type benzodiazepine receptor  
expression and cell proliferation in human breast cancer cells

AU Papadopoulos, Vassilios; Kapsis, Asimina; Li, Hua; Amri, Hakima; Hardwick,  
Matthew; Culty, Martine; Kasprzyk, Philip G.; Carlson, Mark; Moreau,  
Jacque-Pierre; Drieu, Katy

CS Division of Hormone Research, Georgetown University Medical Center,  
Washington, DC, 20007, USA

SO Anticancer Research (2000), 20(5A), 2835-2847  
CODEN: ANTRD4; ISSN: 0250-7005

PB International Institute of Anticancer Research

DT Journal

LA English

RE.CNT 98 THERE ARE 98 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 2000:521260 CAPLUS  
DN 133:207145

TI Ginkgo biloba extract (EGb 761) and CNS functions: basic studies and  
clinical applications

AU DeFeudis, F. V.; Drieu, K.

CS Institute for BioScience, Westboro, MA, 01581, USA

SO Current Drug Targets (2000), 1(1), 25-58  
CODEN: CDTUUA; ISSN: 1389-4501

PB Bentham Science Publishers Ltd.

DT Journal; General Review

LA English

RE.CNT 166 THERE ARE 166 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 1999:57991 CAPLUS  
DN 130:247179

TI In vivo studies on the role of the peripheral benzodiazepine receptor  
(PBR) in steroidogenesis

AU Papadopoulos, V.; Widmaier, E. P.; Amri, H.; Zilz, A.; Li, H.; Culty, M.;  
Castello, R.; Philip, G. H.; Sridaran, R.; Drieu, K.

CS Departments of Cell Biology & Pharmacology, Georgetown University Medical  
Center, Washington, DC, USA

SO Endocrine Research (1998), 24(3 & 4), 479-487  
CODEN: ENRSE8; ISSN: 0743-5800

PB Marcel Dekker, Inc.

DT Journal

LA English

RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
AN 1998:329598 CAPLUS  
DN 129:76458

TI Effect of chronic administration of ginkgo biloba extract or ginkgolide on  
the hypothalamic-pituitary-adrenal axis in the rat

AU Marciilhac, A.; Dakine, N.; Bourhim, N.; Guillaume, V.; Grino, M.; Drieu, K.; Oliver, C.  
 CS Laboratoire de Neuroendocrinologie Experimentale, Institut Jean Roche, INSERM U 297, Faculte de Medecine Ecteur Nord, Marseille, 13916, Fr.  
 SO Life Sciences (1998), 62(25), 2329-2340  
 CODEN: LIFSAK; ISSN: 0024-3205  
 PB Elsevier Science Inc.  
 DT Journal  
 LA English  
 RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
 AN 1997:757955 CAPLUS  
 DN 128:57588  
 TI Ex vivo regulation of adrenal cortical cell steroid and protein synthesis, in response to adrenocorticotrophic hormone stimulation, by the Ginkgo biloba extract EGb 761 and isolated ginkgolide B  
 AU Amri, Hakima; Drieu, Katy; Papadopoulos, Vassilios  
 CS Deps. Cell Biol., Georgetown Univ. Med. Cent., Washington, DC, 20007, USA  
 SO Endocrinology (1997), 138(12), 5415-5426  
 CODEN: ENDOAO; ISSN: 0013-7227  
 PB Endocrine Society  
 DT Journal  
 LA English  
 RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD  
 ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN  
 AN 1997:414183 CAPLUS  
 DN 127:29095  
 TI Ginkgolides for inhibition of membrane expression of benzodiazepine receptors and to combat excess glucocorticoid production  
 IN Drieu, Katy  
 PA Societe de Conseils de Recherches et d'Applications Scientifiques (SCRAS), Fr.; Cockbain, Julian  
 SO PCT Int. Appl., 19 pp.  
 CODEN: PIXXD2  
 DT Patent  
 LA English  
 FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9717068	A1	19970515	WO 1996-EP5005	19961108
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN				
	RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	TW 513305	B	20021211	TW 1996-85113522	19961105
	CA 2235416	AA	19970515	CA 1996-2235416	19961108
	AU 9675717	A1	19970529	AU 1996-75717	19961108
	AU 724416	B2	20000921		
	EP 862428	A1	19980909	EP 1996-938209	19961108
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2000505057	T2	20000425	JP 1997-517873	19961108
	NZ 322050	A	20000728	NZ 1996-322050	19961108
	ZA 9609437	A	19970618	ZA 1996-9437	19961109
	US 6274621	B1	20010814	US 1998-68368	19980723
	US 2002006955	A1	20020117	US 2001-879306	20010612
	US 2005281899	A1	20051222	US 2005-130680	20050517
PRAI	US 1995-7337P	P	19951109		
	US 1995-575902	A2	19951220		
	WO 1996-EP5005	W	19961108		
	US 1998-68368	A1	19980723		
	US 2001-879306	A1	20010612		

OS MARPAT 127:29095

L2 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AN 1996:726043 CAPLUS

DN 126:69966

TI In vivo regulation of peripheral-type benzodiazepine receptor and glucocorticoid synthesis by Ginkgo biloba extract EGb 761 and isolated ginkgolides

AU Amri, Hakima; Ogwuegbu, Stephen O.; Boujrad, Nouredine; Drieu, Katy; Papadopoulos, Vassilios

CS Dep. Cell Biol., Georgetown Univ. Med. Cent., Washington, DC, 20007, USA

SO Endocrinology (1996), 137(12), 5707-5718

CODEN: ENDOAO; ISSN: 0013-7227

PB Endocrine Society

DT Journal

LA English

RE.CNT 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AN 1992:120910 CAPLUS

DN 116:120910

TI Use of platelet-activating factor antagonists as anti-pruritic agents

IN Woodward, David Frederick; Williams, Linda Sue

PA Allergan, Inc., USA

SO PCT Int. Appl., 19 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9118608	A1	19911212	WO 1991-US2003	19910325
	W: AU, BB, BG, BR, CA, FI, HU, JP, KP, KR, LK, MC, MG, MW, NO, PL, RO, SD, SU				
	RW: AT, BE, BF, BJ, CF, CG, CH, CM, DE, DK, ES, FR, GA, GB, GR, IT, LU, ML, MR, NL, SE, SN, TD, TG				
	CA 2083611	AA	19911201	CA 1991-2083611	19910325
	AU 9176908	A1	19911231	AU 1991-76908	19910325
	EP 532512	A1	19930324	EP 1991-907987	19910325
	EP 532512	B1	19961211		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	JP 05507467	T2	19931028	JP 1991-507834	19910325
	JP 3142559	B2	20010307		
	AT 146079	E	19961215	AT 1991-907987	19910325
	ES 2095316	T3	19970216	ES 1991-907987	19910325
PRAI	US 1990-530739	A	19900531		
	WO 1991-US2003	A	19910325		

L2 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2006 ACS on STN

AN 1992:99051 CAPLUS

DN 116:99051

TI Platelet activating factor antagonists interact with GABAA receptors

AU Miller, Lawrence G.; Bazan, Nicolas G.; Roy, R. Beth; Clostre, Francois; Gaver, Anat; Braquet, Pierre

CS Med. Cent., LSU, New Orleans, LA, 70112, USA

SO Research Communications in Chemical Pathology and Pharmacology (1991), 74(2), 253-6

CODEN: RCOCB8; ISSN: 0034-5164

DT Journal

LA English

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NEWS 5 DEC 14 2006 MeSH terms loaded for MEDLINE file segment of TOXCENTER  
NEWS 6 DEC 14 CA/Caplus to be enhanced with updated IPC codes  
NEWS 7 DEC 21 IPC search and display fields enhanced in CA/Caplus with the  
IPC reform  
NEWS 8 DEC 23 New IPC8 SEARCH, DISPLAY, and SELECT fields in USPATFULL/  
USPAT2  
NEWS 9 JAN 13 IPC 8 searching in IFIPAT, IFIUDB, and IFICDB  
NEWS 10 JAN 13 New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to  
INPADOC  
NEWS 11 JAN 17 Pre-1988 INPI data added to MARPAT  
NEWS 12 JAN 17 IPC 8 in the WPI family of databases including WPIFV  
NEWS 13 JAN 30 Saved answer limit increased  
NEWS 14 JAN 31 Monthly current-awareness alert (SDI) frequency  
added to TULSA  
NEWS 15 FEB 21 STN AnaVist, Version 1.1, lets you share your STN AnaVist  
visualization results  
NEWS 16 FEB 22 Status of current WO (PCT) information on STN  
NEWS 17 FEB 22 The IPC thesaurus added to additional patent databases on STN  
NEWS 18 FEB 22 Updates in EPFULL; IPC 8 enhancements added  
NEWS 19 FEB 27 New STN AnaVist pricing effective March 1, 2006  
NEWS 20 FEB 28 MEDLINE/LMEDLINE reload improves functionality  
NEWS 21 FEB 28 TOXCENTER reloaded with enhancements  
NEWS 22 FEB 28 REGISTRY/ZREGISTRY enhanced with more experimental spectral  
property data  
NEWS 23 MAR 01 INSPEC reloaded and enhanced  
NEWS 24 MAR 03 Updates in PATDPA; addition of IPC 8 data without attributes  
  
NEWS EXPRESS FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a,  
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),  
AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.  
V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT  
<http://download.cas.org/express/v8.0-Discover/>  
  
NEWS HOURS STN Operating Hours Plus Help Desk Availability  
NEWS INTER General Internet Information  
NEWS LOGIN Welcome Banner and News Items  
NEWS PHONE Direct Dial and Telecommunication Network Access to STN  
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FILE LAST UPDATED: 6 Mar 2006 (20060306/ED)

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<http://www.cas.org/infopolicy.html>

=> S GINKGOLIDE (P) MAMMARY

626 GINKGOLIDE

81960 MAMMARY

L1 1 GINKGOLIDE (P) MAMMARY

=>

=> D 1 AB

L1 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN

AB The peripheral-type benzodiazepine receptor (PBR) expression and localization correlate with human breast cancer cell proliferation and aggressive phenotype expression. The standardized extract of Ginkgo biloba leaves (EGb 761) and isolated ginkgolide B (GKB) were shown to decrease PBR mRNA expression in adrenal cells. The authors examined the effect of EGb 761 and GKB on PBR expression and cell proliferation in human breast cancer cells. EGb 761 and GKB decreased in a time- and dose-dependent manner PBR expression and cell proliferation in the highly aggressive, rich in PBR, human breast cancer cell line MDA-231 whereas they did not affect the proliferation of the non-aggressive human breast cancer cell line MCF-7, which contains very low PBR levels. This effect was reversible and not due to the antioxidant properties of the compds. tested. Using a human cDNA expression array the authors determined that EGb 761 treatment altered, in addition to PBR, the expression of 36 gene products involved in various pathways regulating cell proliferation. These in vitro data were further validated in an in vivo model where EGb 761 and GKB inhibited the nuclear PBR expression and growth of MDA-231 cell xenografts in nude mice. Taken together, these data suggest that the manipulation of PBR expression could be used to control tumor growth and that EGb 761 and GKB, under the conditions used, exert cytostatic properties.

=> D 1

L1 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2006 ACS on STN

AN 2000:783981 CAPLUS



DN 134:320580  
TI Drug-induced inhibition of the peripheral-type benzodiazepine receptor  
expression and cell proliferation in human breast cancer cells  
AU Papadopoulos, Vassilios; Kapsis, Asimina; Li, Hua; Amri, Hakima; Hardwick,  
Matthew; Culty, Martine; Kasprzyk, Philip G.; Carlson, Mark; Moreau,  
Jacque-Pierre; Drieu, Katy  
CS Division of Hormone Research, Georgetown University Medical Center,  
Washington, DC, 20007, USA  
SO Anticancer Research (2000), 20(5A), 2835-2847  
CODEN: ANTRD4; ISSN: 0250-7005  
PB International Institute of Anticancer Research  
DT Journal  
LA English  
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